

LETTERS TO THE EDITOR

Prognosis in Cardiac Tumors in Infants and Children

The result of the management of the seven cases of cardiac tumors reported by Bini et al. (1) was quite discouraging as only one child survived 3 months. In a recent review of cardiac tumors reported in the English language between 1972 and 1977 (2), I found 47 reported pediatric cases with a survival rate of more than 6 months in 55% of the cases. Forty-four of these tumors were benign with a survival rate of 57%. The survival rate increased significantly if the diagnosis of cardiac tumor was made preoperatively. The reason the survival rate was so low in the series of Bini et al. is probably that their cases were unusually difficult, perhaps because they report from a sophisticated referral center where only the worst cases may be referred. In the seven cases, six tumors were benign, and only one of the patients survived 3 months. Four of the tumors were rhabdomyomas, which appear to have been unusually severe, because all four were associated with tuberous sclerosis. Of the 23 reported rhabdomyoma cases between 1972 and 1977, tuberous sclerosis was present in only approximately 50%. In a less selective case series than that presented from Birmingham, Alabama, the prognosis for infants and children with cardiac tumors appears to be better than that reflected in the article by Bini et al.

In the same issue of JACC, Martin and Boak (3) claim that the diagnosis of cardiac tumors is usually made postmortem. In our review of 263 cases of cardiac tumor reported in 1972 to 1977, 205 (78%) had a preoperative diagnosis that greatly improved the prognosis.

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References

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2. Bogren HG, DeMaria AN, Mason DT. Imaging procedures in the detection of cardiac tumors, with emphasis on echocardiography: a review. *Cardiovasc Intervent Radiol* 1980;3:107-25.
3. Martin JL, Boak JG. Cardiac metastasis from uterine leiomyosarcoma. *J Am Coll Cardiol* 1983;2:383-6.

Ventricular Response to Atrial Fibrillation

With his editorial, Meijler (1) has reopened a controversy that has lain dormant for over 10 years. His studies of the ventricular rhythm in atrial fibrillation using autocorrelation as the crucial measurement have not demonstrated any "patterning" or nonrandom sequences of beat to beat intervals. Therefore, he asserts that

the ventricular rhythm in response to atrial fibrillation is "random," and that certain drugs such as digitalis and quinidine do not affect this property of the ventricular rhythm in atrial fibrillation.

However, studies by several other techniques that are independent of each other demonstrate that the ventricular response to atrial fibrillation in human beings is not random, and instead is patterned in various ways (2-5). The simplest of these consists of casting histograms of ventricular beat to beat intervals that occur during inspiration, expiration and apnea in the presence of quiet, normal respiration and with Cheyne-Stokes respiration. The histograms of ventricular beat to beat intervals during apnea are markedly different from those during expiration, and slightly different from those inscribed during inspiration. Because respiration is a rhythmic process, it is clear that the ventricular response to atrial fibrillation changes in a rhythmic fashion and that the autocorrelogram is simply not sufficiently sensitive to record this rhythmicity.

More important is the fact that many more "patterned" sequences of ventricular beat to beat intervals occur in the majority of patients with atrial fibrillation than would be expected by chance. Our group studied only three types of many possible sequence patterns. These were sequences of equal intervals (intervals that varied no more than 32.5 ms from the first interval of the sequence), sequences of intervals that diminished in duration as expected in Mobitz type I second degree block and sequences of intervals that were multiples of a single common denominator, as expected in varying second degree block (3:2, 3:1, 4:2, etc.). These sequences were located (by computer) in recordings including 7,000 to 15,000 beats (per recording) and classified by the number of beats each sequence contained and by the interval length of their membership. We then calculated from the overall beat to beat interval histogram how many sequences of each type, membership size and interval length would be expected by chance alone in the recording at hand, and compared the number actually found with the number expected. Only calculations based on more than 1,000 intervals with probability (p) values less than 0.001 were called "sequences." Using these rigorous definitions, sequences of patterned intervals were found in most of our patients (6).

In some instances this was so because in the time that it takes to record 7,500 to 15,000 heartbeats, patients with atrial fibrillation may develop paroxysms of atrial flutter, ventricular tachycardia, junctional tachycardia or escape rhythm. However, these nonfibrillatory mechanisms were not the cause of most of the sequences we found. To ascertain this fact, all the sequences were located on the magnetic tape and the scalar electrocardiogram and tachogram reconstituted and then visually inspected. It is clear from these studies and also those by others that the ventricular response in atrial fibrillation is not random. It has also been found that quinidine, digitalis glycosides and other drugs significantly affect the nonrandom character of the ventricular response in atrial fibrillation in human beings.

I agree with Meijler that the mechanism whereby the rhythm in atrial fibrillation is established is still unknown. A detailed discussion of the various possibilities has been presented elsewhere (7). However, there can be no doubt that the integrative properties